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Synergies Between Lactic and Acetic Acids in the Inhibition and Killing of Strains of Candida

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Synergies Between Lactic and Acetic Acids in the Inhibition and Killing of Strains of Candida

Abstract: The most common vaginal diseases are bacterial vaginosis (BV) and vaginal candidiasis (VC), experienced by 29% and 75% of women, respectively. BV is characterized by a depletion of vaginal Lactobacillus species, displaced by dozens of BV-associated species; VC results from high abundance of *Candida albicans* or *C. glabrata*. It is not clear whether vaginal colonization or symptomatic infection by *Candida* is influenced by the vaginal microbiome and whether BV increases risk for VVC. Whether intravaginal *Candida* can flourish may depend on the major acids produced by Lactobacillus (lactate) versus non-Lactobacillus species in BV (acetate). The BV environment is typically pH>5 and <20mM of lactic acid but ~120mM acetic acid, whereas a healthy vaginal environment is pH<4.5, ~120mM lactic acid, and <4mM acetic acid. These combinations are not ideal for inhibiting *Candida*; either the pH is too high, or the acetate is too low. We tested whether combinations of lactic acid and acetic acid inhibit or kill vaginal isolates of *C. albicans* and *C. glabrata*, or whether they alter the antifungal activity of azoles. Preliminary data suggest that acetate and lactate at concentrations characteristic of a healthy vaginal environment inhibit about half of tested *C. albicans* isolates but none of the *C. glabrata* isolates. However, increasing the acetate to 25 mM inhibited all isolates of both species, and the combined acids at optimal concentrations were fungicidal. This suggests that combinations of these acids could supplement standard fluconazole therapy, particularly needed to counter rising azole-refractory responses of VVC by *C. glabrata*.

Keywords: bacterial vaginosis, vulvovaginal candidiasis, candida albicans, candida glabrata, Lactic acid, acetic acid, fluconazole, azoles, infections, metronidazole